ATTEMPTED SUICIDE BY INSULIN INJECTION TREATED WITH HYPERTONIC GLUCOSE SOLUTION

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ABSTRACT

A young woman with no history of diabetes tried to commit suicide by injecting 2800 units of subcutaneous NPH insulin. She was transferred to Loghman Hospital within 12 hours. The main clinical symptom was continuous seizure activity which was resistant to all forms of routine drug therapy. The patient was treated with intravenous hypertonic glucose (50%) followed by continuous glucose (10%) infusion. The patient was discharged from the hospital after 8 days without any sequelae.

Keywords: Insulin, Overdose, Suicide.

INTRODUCTION

Although exogenous hyperinsulinism frequently occurs after accidental injection, it may occur after surreptitious self-injection and occult malicious1 or factitious administration; in addition, many cases of insulin overdose are self-inflicted, often by depressed individuals intent on suicide.2-7 Serious complications after insulin overdose were observed soon after insulin was introduced.8 The incidence of hypoglycemia due to insulin overdose may be more common than is generally appreciated.9 There have been cases of insulin overdose reported in other countries10 but the present patient was the first case referred to Loghman Hospital in the past 15 years.11 In all cases hypoglycemic coma ensues which may be followed by death, hypoglycemic encephalopathy, or recovery depending on how soon normoglycemia is restored.10 Treatment of insulin overdose consists of rousing the patient by means of glucose injection, followed by maintaining consciousness via an adequate intake while taking care to avoid an overload of glucose and insulin deficiency. Although with close medical supervision such treatment is perfectly practical, it is often complicated by rapid and substantial changes in blood glucose concentration.12

Case report

A 21 year old woman with no history of previous insulin use attempted suicide by injecting 2800 units of NPH (isophane insulin, intermediate acting) insulin subcutaneously. As shown in Table I, on arrival she was comatose, agitated, sweating, and showed hypothermia, mydriasis and seizure. There was no sign of trauma on her body. After airway control, oxygenation and intravenous access, 1 vial of 50% glucose solution, 20 mg diazepam and 2 mg naloxone was administered but her clinical presentation did not change.
Suicide with Insulin

Table I. Clinical manifestations, vital signs and blood glucose levels of the patient.

<table>
<thead>
<tr>
<th>Clinical manifestations</th>
<th>Vital signs</th>
<th>Blood glucose level</th>
</tr>
</thead>
<tbody>
<tr>
<td>On admission</td>
<td></td>
<td></td>
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<tr>
<td>Coma, sweating, mydriasis, hyperthermia, convulsion, sinus tachycardia, arrhythmia</td>
<td>Pulse rate 60 Resp. rate: 18</td>
<td>10 min after arrival: 0 mg/dL 30 min after arrival: 5 mg/dL</td>
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<tr>
<td></td>
<td></td>
<td>120/70 Normal</td>
</tr>
<tr>
<td>After administration of hypertonic glucose (5 vials), 6 h post admission</td>
<td>Normal</td>
<td>80-100 mg/dL</td>
</tr>
<tr>
<td>Coma, hypertonia, tremor, mild hyperthermia</td>
<td>Normal</td>
<td>100-120 mg/dL</td>
</tr>
<tr>
<td>48 h post admission</td>
<td>Conscious, nausea, confusion</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Because of her arrhythmias and continuous seizures (unresponsive to diazepam administration), lidocaine (1 mg/kg) and phenytoin (10 mg/kg) were administered which had no significant effect.

Preliminary blood analysis showed 0 mg/dL of glucose. We supposed that there was an error in analysis. The second blood analysis showed a glucose level of 5 mg/dL, this temporary increase being due to 50% glucose administration. Repeat examination confirmed the presence of needle marks on her body. All signs and symptoms were restored after infusion of 5 vials of 50% glucose solution. Particularly the convulsions responded well to glucose administration. Infusion of 10% glucose solution was continued for 5 days, after which oral glucose regimens were begun.

During the first 5 days after admission, her level of consciousness was satisfactory and remained so. After glucose infusion for 5 days and oral intake for 3 days, the blood glucose concentration continued to be stable between 100-120 mg/dL though no more glucose was required after 8 days of admission, at which time she was discharged from the hospital.

DISCUSSION

The typical patient with an insulin overdose is often found in an unconscious state. Patients with intermediate or extended action insulin overdose may not develop symptoms for 18 to 36 hours (coma, vomiting, etc.). Mortality in attempted suicidal overdose with insulin is 25%. Five of 20 diabetics who took 800 to 3200 U of insulin died and there was little correlation between insulin dose and severity of hypoglycemia. Our data are not sufficient to prove this subject.

The treatment of a patient who has taken excessive doses of insulin is to maintain normoglycemia, as hypoglycemia deprives the brain of substrate for oxidation and causes cerebral damage similar to that caused by hypoxia. Intravenous glucose with glucagon is the traditional method of treating hypoglycemia and if cerebral edema occurs dexamethasone and mannitol may be used. Intramuscular glucagon, 1 to 2 mg, has a fast onset of action and mobilizes hepatic glycogen stores; however, once hepatic glycogen stores are exhausted (prolonged hypoglycemia, fasting, alcohol abuse), the effect may be variable or nil. Unfortunately, glucagon was not administered in this case, because it was not available. Glucose administration may be required for more than 5 days before the effect of insulin overdosage is overcome (as demonstrated by a glucose concentration >100 mg/dL on two successive occasions). We had to administer glucose for more than 5 days in this case (Table I). Fortunately no neurological sequelae remained and the patient was treated successfully. Reports suggest that the period from injection of an overdose of insulin to irreversible brain damage is frequently about 7 hours.
This case is the first insulin intoxication which we have encountered during the past 15 years, and once again emphasizes the importance of exact and precise patient examination in identifying uncommon routes of poisoning.

REFERENCES
